

REPORT DOCUMENTATION PAGE

AD-A218 839

2b. DECLASSIFICATION / DOWNGRADING SCHEDULE

1b. RESTRICTIVE MARKINGS

None

3. DISTRIBUTION / AVAILABILITY OF REPORT

Approved for public release; distribution is unlimited

4. PERFORMING ORGANIZATION REPORT NUMBER(S)

USAFSAM-JA-89-47

5. MONITORING ORGANIZATION REPORT NUMBER(S)

6a. NAME OF PERFORMING ORGANIZATION

USAF School of Aerospace Medicine

6b. OFFICE SYMBOL

(If applicable)

USAFSAM/HM

7a. NAME OF MONITORING ORGANIZATION

6c. ADDRESS (City, State, and ZIP Code)

Human Systems Division (AFSC)
Brooks Air Force Base, TX 78235-5301

7b. ADDRESS (City, State, and ZIP Code)

8a. NAME OF FUNDING / SPONSORING ORGANIZATION

USAF School of Aerospace Medicine USAFSAM/HM

8b. OFFICE SYMBOL

(If applicable)

9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER

8c. ADDRESS (City, State, and ZIP Code)

Human Systems Division (AFSC)
Brooks Air Force Base, TX 78235-5301

10. SOURCE OF FUNDING NUMBERS

PROGRAM
ELEMENT NOPROJECT
NO.TASK
NOWORK UNIT
ACCESSION NO

SUPT

XX

HM

11. TITLE (Include Security Classification)

Treatment of Methylene Chloride Induced Carbon Monoxide Poisoning with Hyperbaric Oxygenation

12. PERSONAL AUTHOR(S)

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13a. TYPE OF REPORT

Final

13b. TIME COVERED

FROM TO

14. DATE OF REPORT (Year, Month, Day)

15. PAGE COUNT

16. SUPPLEMENTARY NOTATION

17. COSATI CODES

FIELD

GROUP

SUB-GROUP

06

05

06

11

18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)

Carbon monoxide; Methylene chloride; Hyperbaric oxygen

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

Methylene chloride is an organic solvent with many industrial uses. Inhalation of methylene chloride fumes can result in toxicity, caused by hepatic biotransformation of methylene chloride to carbon monoxide. A case of acute methylene chloride poisoning is presented, including successful treatment of this patient with the use of hyperbaric oxygenation. The rationale for the use of hyperbaric oxygenation in the treatment of methylene chloride poisoning is discussed.

20. DISTRIBUTION / AVAILABILITY OF ABSTRACT

☒ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT ☐ DTIC USERS

21. ABSTRACT SECURITY CLASSIFICATION

Unclassified

22a. NAME OF RESPONSIBLE INDIVIDUAL

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22b. TELEPHONE (Include Area Code)

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22c. OFFICE SYMBOL

USAFSAM/HM

Number pages 7
Word count 1967
Tables/figures 0
Phone 512-536-3281

TREATMENT OF METHYLENE CHLORIDE INDUCED CARBON MONOXIDE
POISONING WITH HYPERBARIC OXYGENATION

Running head: HBO and methylene chloride poisoning

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Key terms:

Methylene chloride
Carbon monoxide
Hyperbaric oxygen

ABSTRACT OF

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TREATMENT OF METHYLENE CHLORIDE INDUCED CARBON MONOXIDE POISONING WITH HYPERBARIC OXYGENATION

Introduction

Each year in the United States, over 70,000 workers are exposed to methylene chloride (1). Methylene chloride (CH_2Cl_2), a simple halogenated hydrocarbon, is a colorless, highly volatile solvent with many industrial uses, including use as a paint stripper, degreaser, aerosol propellant, and as a solvent for cements and plastics (2). It has been described as being low in toxicity (3), and was previously thought to be inert in vivo (4). In 1972, Stewart et al (5) noted that inhalation of concentrations of methylene chloride presumed to be "safe" resulted in prolonged periods of carboxyhemoglobinemia, which continued to rise after the exposure was ended. Methylene chloride is highly lipid soluble, and is deposited in a variety of body tissues, from which it is slowly released (6). In the liver, mixed function oxidases convert methylene chloride to carbon dioxide and carbon monoxide (7). In this paper, a case of methylene chloride induced carbon monoxide poisoning is presented, followed by a discussion of the treatment of carbon monoxide poisoning with the use of hyperbaric oxygen therapy.

Case Report

A 25-year-old white male presented to his local hospital emergency room complaining of severe headache, nausea, and incoordination. One day prior to presentation he was cleaning computer equipment with methylene chloride, performing this activity for 6 - 8 hours without adequate ventilation or the use of gloves. He had not used this type of solvent previously. He denied a history of smoking or recent ethanol use. He felt well throughout the day, but noted mild headache and dizziness upon retiring that night. The following morning he awakened with a severe headache and nausea, and vomited several times. He also noted

incoordination, including inability to drive his car, and sought medical attention.

Vital signs in the emergency room showed temperature 97.6 F orally, pulse 100 bpm, respirations 20/min, and blood pressure 140/80 mmHg. He was fully awake, coherent, and in no distress. Head and neck examinations were normal. Chest was clear bilaterally, and heart had a regular rate and rhythm with no murmurs. Abdominal and neurological examinations were normal. Chest X-ray was normal, as were liver function tests, CBC, electrolytes, and urinalysis. Arterial blood gas (on 100% O₂) showed pH 7.39, pCO₂ 33, pO₂ 298, HCO₃ 21. Initial COHb was 20.1.

The patient was continued on 100% O₂ by mask, and arrangements were made for hyperbaric oxygen therapy. The patient received hyperbaric oxygen (HBO) therapy via the USAF carbon monoxide treatment table, consisting of two 23-minute oxygen breathing periods at 3 ATA (66 FSW), followed by two 25-minute oxygen breathing periods at 2 ATA (33 FSW). The dive was tolerated well, with no problems noted. Following a period of observation, he was discharged home, and remained asymptomatic.

Discussion

Carbon monoxide poisoning is the most common type of poisoning, with thousands of cases each year, many of which are unrecognized (8). Carbon monoxide usually results from inhaling the product of incomplete combustion of hydrocarbons, from such sources as automobile exhaust, structural fires, and from space heaters.

The toxic effects of carbon monoxide derive from a number of mechanisms. Haldane noted that carbon monoxide binds reversibly to hemoglobin, and can be displaced by the use of high pressures of oxygen (9). Experiments by Goldbaum et al (10) have demonstrated that the toxic effects of CO are independent of its interaction with hemoglobin. It is now known that CO binds to other heme compounds, including myoglobin (11), cytochrome oxidase (12), and P-450 (13).

A form of carbon monoxide poisoning that has received relatively less attention in the literature is the endogenous formation of carbon monoxide following toxic exposure to methylene chloride. Horowitz (14) described a

case of carboxyhemoglobinemia induced by methylene chloride inhalation (paint sniffing), treated with 100% oxygen. He speculated that hyperbaric oxygenation might be of benefit in these cases. Youn et al have reported their experience at the Maryland Institute of Emergency Medical Services in treating 12 cases of methylene chloride poisoning caused by a single spill (15). Nine of the patients were treated with hyperbaric oxygenation, with excellent results.

A very important difference exists between treatment of poisoning from inhaling CO and poisoning from endogenous production of CO from methylene chloride exposure. In the former, tissue CO levels have peaked, and begin to fall, after the exposure is terminated. In methylene chloride induced CO poisoning, CO levels continue to rise after the exposure is terminated, as endogenous production by the liver continues. As a result, patients adequately treated soon after their exposure may develop a recurrence of symptoms which may require retreatment. A period of observation (12 - 24 hours post-exposure) is required after methylene chloride poisoning to ensure complete resolution.

Based on the widespread use of this solvent, it is surprising that more cases of poisoning are not reported. Possibly many such cases go unrecognized. Therefore, physicians should be aware of the signs and symptoms of carbon monoxide poisoning, including headache, nausea, and neurologic disturbances. Poisoning with methylene chloride must be considered when patients presenting with signs of carbon monoxide poisoning have a history of recent exposure to solvents, or when the carboxyhemoglobin level continues to rise despite removal of the exposure source. Treatment should consist of supportive measures, administration of 100% oxygen, and with the use of hyperbaric oxygenation whenever available.

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